Stress and Health

Michele M. Larzelere, PhD[a,*], Glenn N. Jones, PhD[b]

KEYWORDS

• Stress • Stress reduction • Meditation

Stress is any situation in which environmental or perceived demands force significant psychologic or biological change upon an organism, to preserve homeostasis or ensure survival. Stress results from physical or psychosocial disequilibrium. Stress responses are those behavioral, psychologic, or physiologic efforts to compensate for situational demands. Stress responses sometimes lead to increased disease risk.

It often can be useful to conceptualize an individual's reaction to stress in accordance with the general adaptation syndrome, as proposed by Selye.[1] Upon being presented with a physical or emotional stressor, the individual recognizes (alarm reaction) and initially mounts a strong physiologic (or psychologic or behavioral) reaction to the stressor (stage of resistance) until such time as the challenge is met, the stressor has passed, or the organism's ability to mount the response is depleted (stage of exhaustion). This model is understood easily by patients and allows one to draw explicit attention to the physiologic and psychologic costs of fighting a stressor. This paradigm easily adapts to situations in which multiple stressors, chronic stressors, or personal or environmental factors decrease an individual's coping abilities.

Stress can be categorized in several ways, including by duration (acute/chronic), domain (physical/psychologic), and severity (traumatic[†]/daily hassles). Although physical strain often is documented and quantified, psychologic stress can be more difficult to define. Psychologic models of stress rely on the concept of perceived stress (eg, events or situations are only stressful to the degree that the individual defines them as straining his or her ability to cope). The appraisal of the situation as threatening brings about the physiologic and behavioral changes defined

[a] Department of Family Medicine, Louisiana State University Health Sciences Center–New Orleans, 200 West Esplanade Avenue, Suite 412, Kenner, LA 70065, USA
[b] Department of Family Medicine, Louisiana State University Health Sciences Center, Earl K. Long Medical Center, 5825 Airline Highway, Baton Rouge, LA 70805, USA
* Corresponding author. Department of Family Medicine, Louisiana State University Health Sciences Center–New Orleans, 200 West Esplanade Avenue, Suite 412, Kenner, LA 70065.
E-mail address: mlarze@lsuhsc.edu (M.M. Larzelere).

[†] A full description of the nature and treatment of post-traumatic stress disorder (PTSD) is beyond the scope of this article, so the remainder of this discussion will be devoted to the health implications and coping challenges inherent to stress of a nontraumatic nature. Readers interested in a comprehensive treatment of PTSD are referred to *Handbook of PTSD*: *Science and Practice*, edited by MJ Friedman, TM Keane, and PA Resick. New York: Guilford; 2007.

Prim Care Clin Office Pract 35 (2008) 839–856
doi:10.1016/j.pop.2008.07.011 **primarycare.theclinics.com**
0095-4543/08/$ – see front matter

as the stress response. The controllability of a stressor also significantly impacts its potential for long-term psychologic detriment. Stress-causing situations do not necessarily have to be negative. Selye[1] chose the term *adaptation*, noting that both pleasant and unpleasant events can evoke the stress response. Similarly, Holmes and Rahe[2] focused on change as a stress in their pioneering studies. Their Social Readjustment Rating Scale asks about several life changes, some of which might be pleasant (vacation) or positive (promotion). Across this variety of stressor types, research has documented the possibility of resultant negative emotional and physical impacts.

THE IMPACT OF STRESS ON PHYSICAL FUNCTIONING

The fight-or-flight response to acute stress causes rapid changes in the nervous, cardiovascular, immune, and endocrine systems. Cortisol and catecholamines are produced to increase energy availability. Heart rate and stroke volume are increased. The immune system is activated to prepare for the possibility of injury. Less vital activities (eg, feeding, growth, reproduction) are suspended during the crisis. Many of these changes have physiologic costs that are minimized by a rapid return to homeostatic baseline following the cessation of the stressor. Acute stressors in healthy adults are unlikely to have negative impacts on health. Stressful situations often persist beyond the time period when these physiologic coping strategies are adaptive, however. Further, psychologic factors lead some individuals to turn acute stressors into chronic stressors because of their meanings or implications. In addition, physiologic response magnitude differs between individuals because of genetic influences and previous stress exposures, which cause some individuals to produce a sustained hyper-response to stress.

Stress and the Endocrine System

The corticotropin-releasing factor system is the integrator of the brain's (central nervous system [CNS]) response to stress and negative emotion. Under conditions of stress, cells of the hypothalamus control the secretion of corticotropin releasing hormone (CRH), which stimulates release of ACTH (adrenocorticotropic hormone). ACTH activity leads to the secretion of glucocorticoid hormones (cortisol) from the adrenal cortex. This constitutes the hypothalamic-pituitary-adrenal (HPA) axis. Glucocorticoids provide inhibitory feedback on the HPA that helps to limit the duration of the stress response. The sympathetic adrenal medullary (SAM) axis often acts in parallel with the HPA axis. CRH stimulation of sympathetic nervous system activity also leads to the release of epinephrine and noradrenaline.

The interactions of adrenal system components under conditions of stress are nonlinear.[3] Some of the stress-/cortisol-related changes include suppression of gonadotropin-releasing hormone and inhibition of thyroid-stimulating hormone (TSH) release. Although acute stress may increase plasma concentrations of growth hormone (GH), chronic activation of the HPA axis inhibits growth through suppression of GH secretion and inhibition of the action of GH on target tissues. Chronic glucocorticoid elevations may cause individuals to experience catabolic effects (visceral adiposity, decreased lean body mass) and increased insulin resistance. This can lead to increased difficulties achieving glycemic control in diabetic patients under stress.

Cortisol also plays a role in memory formation by means of its role on the amygdala and hippocampus. Elevated cortisol levels, during conditions of stress, may aid the formation of emotionally valenced long-term memories, promoting enhanced response to similar future stressors. This sensitization of the amygdale to further